A DISSERTATION

ON

RIGHT VENTRICULAR INFARCTION – A CLINICAL STUDY

Submitted to
THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY
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CERTIFICATE

This is to certify that the dissertation entitled **"Right Ventricular Infarction – A Clinical Study"** is a bonafide work done by **Dr. K. VENKATESAN** in **M.D BRANCH I GENERAL MEDICINE** at Government Mohan Kumaramangalam Medical College, Salem, to be submitted to The Tamil Nadu Dr.M.G.R Medical University, in fulfillment of the University Rules and Regulation for the award of M.D. Degree Branch I General Medicine, under my supervision and guidance, during the academic period from January 2006 to July 2007.

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DECLARATION

I solemnly declare that this dissertation **"Right Ventricular Infarction – A Clinical Study"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem under the guidance and supervision of **Prof. Dr. R. NATARAJAN, M.D.,** Unit Chief of General Medicine, Govt. Mohan Kumaramangalam Medical College and Hospital Salem.

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CONTENTS

INTRODUCTION

Myocardial infraction is the term used when the myocardium is necrosed due to ischemia. It may be transmural or subendocardial.

Inferior wall infarction has got some special features like association with right ventricular infarction and brady arrhythmias especially sinus bradycardia and second degree AV Block.

Right ventricular infarction is different from that of the left ventricle in the acute presentation, therapy and long term prognosis.

The early recognition of Right ventricular myocardial infarction is important, because the time of onset of its hemodynamic consequence is unpredictable and these may be prevented by the administration of intravenous fluid load.

The description of Right ventricular myocardial infarction appeared more than 60 years ago. But it was considered unimportant until Cohn and co-workers in 1974 published their classic report on Right ventricular myocardial infarction as a distinct clinical entity.

The reported incidence is between 25-50% of Inferior Wall Myocardial Infarction.

Involvement of RV s related to severe altherosclerotic occlusion of the right coronary artery and is associated with involvement of postero-inferior wall and posterior of the septum.

Clinically Right ventricular myocardial infarction can be suspected when a patient with Inferior Wall Myocardial Infarction presents with elevated JVP, positive kussmaul's sign, hypotension, right sided third or fourth hear sounds, tender hepatomegaly, oliguria, rarely TR and clear chest.

Electrocardiogram was generally believed to be unhelpful in identifying Right ventricular myocardial infarction, until Erhardt and co-workers described the value of a right precordial lead in patient with autopsy proven Right ventricular myocardial infarction. A 1 mm ST elevation in this lead is 70% sensitive and 100% specific. The change is transient. In one series, 48% of the patients had resolution of ECG changes with in 10 hours of the onset of symptoms.

Since bradycardia is the most common arrhythmia occurring during early phases of Acute Myocardial Infarction and is particularly frequent in patients with inferior and posterior infarction.

All types of AV conduction disturbances and intraventricular blocks can occurs more commonly in Inferior Wall Myocardial Infarction than AWMI. Of these, 2nd mobitz type – 1 AV block occurs commonly in Inferior Wall Myocardial Infarction than Anterior Wall Myocardial Infarction. This is usually transient and does not persist for more than 72 hours after infarction.

AIM OF THE STUDY

To study the clinical profile of Right ventricular myocardial infarction and,

- To find the incidence of Right ventricular myocardial infarction in patients having inferior wall myocardial infarction.
- To analyse age and sex distribution
- To analyse the symptamatology and risk factors.
- To analyse the clinical features.
- To analyse the complication and outcome following Right ventricular myocardial infarction.
- To analyse the mortality following right ventricular myocardial infarction.

REVIEW OF LITERATURE

William Harvey, in 1667 was perhaps the first to report post mortem findings correlated with clinical description in a patient with ischaemic heart disease. His subject, a middle aged man, had numerous episodes of “distressing pain in the chest” and died during one of them. The autopsy revealed rupture of the left ventricle. A somewhat similar patient was reported by Thomas Willis in 1674, but the observation of Harvey and Willis seems to have attracted little attention. No more did the report of morgagni who, in his de sidibus et causes morborum published in 1761, citerd an autopsy performed in 1743. The left coronary artery of the patient who had experienced “a speedy death” “appeared to have changed into a bony canal”.

Heberden’s description of angina pectoris in 1772 may be considered as the beginning of modern period in the history of CHAD. His clinical description was so accurate that little has been added subsequently.

In 1842, Marshall Hall emphasized that experimental obstruction of a coronary artery was soon followed by death. In 1896, George Dock reported a patient with severe chest pain and pericardial

friction rub whose clinical diagnosis of coronary thrombosis with myocardial infarction and pericarditis was confirmed at autopsy.

James B. Herrick in (1912) described the clinical picture of obstruction of coronary arteries. Herrick`s delineation of the clinical picture was so clear that it now became possible for other physicians to suspect and diagnose it correctly. Nevertheless the first decade after his paper was, in general, a period of resounding silence, despite further reports from Herrick and an experimental study by Fred Smith who is one of his pupils demonstrating typical electrocardiographic changes associated with infarction of heart.

Most medical students and house officers of the 1920-25 era never heard of the disease during their period of clinical training. Several more years were to elapse before myocardial infarction was generally recognized. Subseuent history of myocardial infarction is that of advances in diagnostic accuracy and improvements in treatment.

Initially the diagnosis of right ventricular infarction was at autopsy. In 1948, Wartman & Hallerstein, described 22 instances of Right ventricular myocardial infarction out of 164 cases of myocardial

infarction they autopsied". In 1959 Wade described 19 patients with right ventricular infarction.

Even though the description of right ventricular infarction appeared more than 60 years ago it was not considered as an important clinical entity, in large part because of studies in animals in which experimentally induced isolated right ventricular damage caused no substantial change in systemic venous pressure, pulmonary pressure or cardiac output.

In 1974, Cohn and co-workers published their classic report on right ventricular infarction as a distinct clinical entity.

Typically right ventricular infarction occurs with occlusion of RCA proximal to acute marginal branches. Occasionally occlusion of a dominate let circumflex may also produce Right ventricular myocardial infarction.

LAD occlusion may result in the infarction of the anterior right ventricle.

Isolated right ventricular infarction accounts for less than 2% percent of all cases of cases of infarction but may result in considerable morbidity. It is associated with,

1. Isolated occlusion of right ventricular branches.
2. Occlusion of non-dominant right coronary artery and
3. Occlusion of dominant right coronary artery with good collateral flow to posterior descending artery. But it may occur in the absence of coronary disease when substantial right ventricular hypertrophy is present (eg COPD, PS, Pulmonary hypertension, etc).

MAJOR SECTIONS OF THE RIGHT CORONOARY ARTERY WITH CORRESPONDING REGIONS OF PERFUSION AND ECG FINDINGS FOR UNDERPERFUSION

Arterial Segment	Arterial Branch	Perfused region	ECG effects of ischemia
Proximal Segment	SA Nodal branch Righ atrial branch	SA Node Atrial free wall	Sinus bradycardia Atrial infarct pattern, atrial fibrillation
Middle Segment	Lateral RV branches Marginal RV branch	**Lateral RV free wall** Inferior (Posterior) RV free wall	ST segment elevation; later, abnormal Q waves in leads V_3R through V_6R
Distal Segment	AV nodal branch	**AV node**	AV block
Posterior descending	Posterior lateral LV branches	**Posterior lef ventricle**	ST segment elevation; later,

segment	Posterior descending artery	**Inferior septum, inferior LV free wall**	abnormal Q waves in leads II, III and aVF
ECG = electrocardiographic; SA = sinoatrial; RV = Right ventricular; AV = atrioventricular; LV = left ventricular.			

Ischemic injury can produce conduction block at any level of the AV or intraventricular conducting system.

First degree AV Block occurs in less than 15 percent of patients with acute myocardial infraction admitted to CCUs.

Mobitz type I block occurs in upto 10% of acute myocardial infarction. It occurs more commonly in inferior than anterior myocardial infarction. Mobitz type II block is rare following acute myocardial infarction accounting for 10% of all cases of second degree heart block. Complete heart block may occur in 5-15 percent of patients with either anterior or inferior wall myocardial infarction. The incidence is higher in patients with right ventricular infarction. Bundle branch blocks are reported to occur in 5-10 percent of patients with acute myocardial infarction.

The usual setting will be therefore the patient that presents with an Acute Inferior Wall Myocardial Infarction. 30-50% if these

patients may have som R.V. involvement although it will only be haemodynamically significant in a minority.

ANATOMY OF CORONARY CIRCULATION

The coronary arteries, the right and the left are the first branches of the aorta. The left coronary artery divided into the left anterior descending and left circumflex artery supplies the left atrium and parts of posterior and lateral wall of the left ventricle. In 35% of cases this artery supplies the S.A. node and in 20% of cases the A.V. node as well.

The right coronary artery supplies the whole of right ventricle, variable parts of diaphragmatic aspect of left ventricle, the posteroinferior third of the interventricular septum, the right atrium and part of the left and the conducting systems as far as proximal parts of right and left bundle branches. Right coronary artery supplies AV node in 80% and SA node in 65% of cases. In 50% of cases right atrium is supplied by right coronary artery and the remaining have dual supply. The left atrium is supplied by the left coronary artery in 62% and right coronary artery in 27% if cases.

The venous blood from heart is drained into right atrium by the coronary sinus, thebesian veins and the anterior cardiac veins.

Atheromatous occlusion of right coronary artery is as common as that of left coronary artery.

ANATOMICO-PATHOLOGICAL CORRELATION

Right ventricular myocardial infarction occurs due to proximal occlusion of the dominant right coronary artery.

Rarely isolated occlusion of right ventricular branches of right coronary artery ca produce infarction of anterolateral aspects of right ventricle alone.

Occlusion of posterior descending artery produces infarction of posterior wall of right ventricle associated with infarction of posteroinferior wall of left ventricle and posterior part of interventricular septum.

In 90% of cases posterior descending artery is a branch of right coronary artery. Therefore, if the collateral to posterior descending artery is poor and right coronary artery is the dominant artery,

occlusion of main stem of right coronary artery produces infarction of both anterolateral and inferior aspects of right ventricle and if those territory supplied by posterior descending artery is good, its territory may escape. In 10% of cases posterior descending artery is an extension of left circumflex artery also called left dominance. In this situtation infarction of posterior wall of right ventricle can occur with a normal right coronary artery.

Thus right ventricular infarction is always associated with infarction of one or the other wall of left ventricle. Right ventricle infarction can occur alone in the following situations.

1. Isolated occlusion of right ventricular branches.
2. Occlusion of Non-dominant right coronary artery and
3. Occlusion of dominant right coronary artery with good collateral flow to posterior descending artery.

In nearly 60% of population the sino atrial node artery arises from right coronary artery and supplies sino atrial node and right atrium. Occlusion of right coronary artery before origin of this branch is likely to cause ischemic damage to right atrium and SA node. Atrioventricular node artery is a branch of posterior descending artery.

Ischemic dysfunction of this node therefore, occur only when blood flow in posterior descending artery is compromised. In cases of occlusion of right coronary artery. A.V. node can escape if there is good collateral flow to the posterior descending artery or if this vessel is arising from circumflex artery.

PATHOPHYSIOLOGY OF Right ventricular myocardial infarction

- The right ventricle has the same cardiac output as the left
- RV is anatomically and physiologically designed to serve the low pressure pulmonary circulation.
- Muscle mass RV is only 15% of LV
- RV stroke work is 25% of LV
- Pulmonary is 10% of Systemic vascular resistance
- Coronary blood flow occurs in both systole and diastole in the right ventricle in the absence of right ventricular hypertrophy.
- The main blood vessel is the right coronary artery (serves the lateral wall the posterior wall and posterior interventricular septum by the pose descending a).
- The anterior wall is supplied by the conus artery and the LAD.

- The RV is like a pocket, wrapped around the LV, sharing the interventricular septum and pericardium.
- The lower after load and myocardial oxygen demand of the right ventricle (as compared with the left) explain its lower oxygen extraction at rest and its relative resistance to irreversible ischemic damage during right coronary occlusion.
- The increased incidence of right ventricular infarction in patients without a history of preinfarction angina may result from a lack of adequately formed collateral vessels.
- Right ventricular infarction complicates 30-50% of inferior wall MI, and 10% of anterior wall infarcts.
- The most reliable ECG finding is ST segment elevation in the right precordial leads, particularly RV4, with associated ST segment elevation in II, III& aVF.
- Clinical signs are: high central venous pressure, clear lung fields and systemic hypotension.
- With a large infarction, the RV may, essentially, become a conduit from the systemic veins to the pulmonary circulation.

- Management is centered on volume loading to CVP 16-20 and the early use of inotropes (dobutamine) to maintain blood flow to the LV (preload) and to maximize cardiac output.
- Right ventricular infarction occurs when there is an occlusion of the right coronary artery proximal to the acute marginal branches, but it may also occur with an occlusion of the left circumflex artery in patients who have left dominant coronary circulation.
- Right ventricular myocardial infarction less commonly may occur as a result of occlusion of the LAD.
- Right ventricular myocardial infarction is strongly associated with inferior wall myocardial infarcts – 30%
- Haemodynamic insufficiency in the presence of inferior wall myocardial infarction suggestions additional right ventricular infarction.
- Isolated Right ventricular myocardial infarction is extremely rare – 2% of autopsies.

HAEMO DYN Acute Myocardial Infarction**CS**

- Hemodynamic sequelae include : Raised Right atrial pressure, Right atrial end diastolic pressure, normal LVEDP.
- “Backward Failure – raised JVP
- Ischaemia iduced RV systolic failure and enlargement of the RV, the restraining effect of the pericardium leads to in left ventricular size and consequent reduced LV performance.
- Remember that normally the tension developed with the contraction of the left ventricle is transmitted to the RV and this assists in the propulsion of blood into the pulmonary arteres.
- In RV infarction, this is less effective:
- Due to LV dysfunction
- Due to flaccidity of the RV free wall and a reduction in it’s contribution to contractility.
- As a result, it is vital to maintain LV preload and RV afterload to minimize LV dysfunction.

Because of the pathophysiology of right ventricular infarction, its management differs substantially from the routine management of left ventricular infarction. Early, accurate diagnosis is imperative.

Since hemodynamically important right ventricular infarction typically occurs in patients with an acute inferior myocardial infarction, suspicion is warranted in any patient presenting with such an infarction.

Clinical Diagnosis:

- The clinical triad of hypotension, clear lung fields, and elevated jugular venous pressure in a patient with an inferior infarction is virtually pathognomonic for right ventricular infarction. However, this triad has a sensitivity of less than 25 percent.
- Caution must be exercised in relying on such findings, since they are readily masked by volume depletion and because the physical and hemodynamic signs of right ventricular infarction often emerge only after volume loading.

Criteria for Acute Ischemic Right Ventricular Dysfunction

- Right-sided (leads V_3R through V_6R) ST segment elevation of greater than 1 mm.
- Right ventricular asynergy as demonstrated by echocardiography or cardiac nuclear imaging.

- Mean right arterial pressure of 10 mm Hg or greater, or a less than 5 mm Hg difference from mean pulmonary capillary wedge pressure (equivalent to left atrial pressure)
- Noncompliant right atrial pressure waveform pattern (steep and deep x and y descents).

INVESTIGATIONS

ECG changes of Right ventricular infarction:

It has been shown that right ventricular infarction can be diagnosed accurately from surface ECG. The electrocardiographic diagnosis of right ventricular infarction is usually based on the manifestation of an elevated ST segment in the extreme right leads V3R and V4R.

Right ventricular infarction should be strongly suspected if, in the setting of acute inferior wall infarction, there is ST segment elevation of 1mm or more in lead VI, lead V4R or any one of the extra right precordial leads, V_4R-V6R. 49-33.

According to Braat et al, an ST segment elevation of more than or equal to 1mm in lead V4R in patient with inferior wall infarction,

had a sensitivity of 100% specificity of 87% and predictive accuracy of 92% in the diagnosis of right ventricular infarction.

The ST segment which is higher in lead V4R than in lead VI and V3R offer the highest specificity and efficiency in diagnosis. 50 These changes disappeared within 10-18 hrs after the onset of chest pain in 50% of their patients.

Lewis and associates in an interesting study of inferior wall infarction, found that an ST segment in lead V2 which is 50% or less than the magnitude of the ST segment elevation in lead a VF indicates right ventricular ischemic injury. Occasionally right ventricular infarction may be associated with ST segment elevation in lead V1 and other precordial leads. But the magnitude of elevation comes down from V1-V5.

I one series 48% of the patients had resolution of ECG changes within 10hrs of the onset of symptoms. Thus it is imperative to record the ECG through the accessory right precordial leads as early as possible.

It is important to recognize the transient nature of ST segment elevation. A right precordial lead V4R is the investigation of choice. ST segment elevation in lead V4R remains the most predictive ECG finding for Right ventricular myocardial infarction.

Right Bundle Branch Block and complete heart block are the most frequent conduction abnormalities associated with Right ventricular myocardial infarction.

USE of 18-LEAD electrocardiogram in Diagnosing Right Ventricular and Posterior wall Involvement in Patients with Acute Inferior Myocardial Infarction.

Acute inferior myocardial infarction (MI), in complicated by right ventricular (RV) infarction, is associated with increased in-hospital morbidity and mortality. The detection of RV and posterior wall (PW) involvement is important and is made possible by direct interrogation of right ventricle using right-sided chest leads (i.e. RV4-6) and PW using leads V7-9 (V7-left posterior axilary line, V8 – inferior angle of left scapula, V9-left paravertebral area. ST elevation

in lead III greater than II of at east 1mm on the standard 12-lead electrocardiogram (ECG) has been shown to have high sensitivity and positive predicative value of RV infarction.

Enzyme Study

Measurement of enzymes released to circulation by irreversibly damaged myocardial cells helps to detect Acute Myocardial Infarction. Serum creatinine kinase (CK) level is the earliest to rise, detectable within 4-8 hours after onset of Acute Myocardial Infarction, reaches a peak in 24 hrs and declines to normal within 3-4 days after the onset of pain. Of the three isoenzymes CK-MB remains the most useful test for myocardial necrosis.

The LDII level starts rising by 24-48 hrs after Acute Myocardial Infarction, reaches a peak in 3-6 days after the onset of MI and returns to normal levels 8-14 days after the infarction.

SGOT starts rising 8-112 hours after the onset of pain, peaks at 18-36 hrs and generally comes back to baseline in 3-4 days.

New Serum Markers for Myocardial Infarction

Cardiac Troponin – T (cTnT) & Cardiac Troponim I (cTnI). This is released to circulation when there is loss of integrity of myocardial cell membrane along with other proteins of cardiac contractile apparatus. The presence of cTnT in the serum has been found to be 100% sensitive for the diagnosis of Acute Myocardial Infarction. It has a long diagnostic window (it can be detected upto 12days post infarction). So it has a role for the late diagnosis of Acute Myocardial Infarction. It is also useful for monitoring response to reperfusion therapy.

Myoglobin: **This is first serum marker to increase in serum after Acute Myocardial Infarction. But it is less specific for myocardium and increased levels can be present in muscular injury or renal disease.**

Echocardiography:

- Two-dimensional echocardiography: abnormal findings include right ventricular dilatation, right ventricular wall asynergy, and abnormal inteventricular septal motion caused by a reversal of the transseptal pressure gradient due to the increased right ventricular end-diastolic pressure.
- The short-axis view has been shown to have the highest sensitivity (82 percent), with a specificity ranging from 62 percent to 93 percent for hemodymnamically important right ventricular infarction.
- Interatrial septal bowing toward the left atrium, indicative of an increased right atrial-left atrial pressure gradient, is an important prognostic marker in right ventricular infarction. Patient with this finding have more hypotension, more heart block and higher mortality than patients without it.
- Doppler echocardiography is particularly helpful in detecting such complications of right ventricular infarction as tricuspid regurgitation, ventricular septal defect and premature opening of the pulmonary value (which indicates a noncompliant right ventricle).

MANAGEMENT

The mortality from Acute Myocardial Infarction halved after the introduction of CCU's and about 40% of the fall in mortality is caused by such interventions as coronary care units, pre-hospital resuscitation and new mechanical and medical treatments of coronary artery disease.

Pain Relief

Alleviation of pain is a critical factor in the case of patients with Acute Myocardial Infarction, and morphine is the drug of choice. Morphine should be used cautiously in Right ventricular myocardial infarction.

If hypotension is not there, sublingual nitrates are given and for patients with waxing and waning pain nitroglycerine is given intravenously. Niroglycerine infusion should be given with extreme caution in patients with acute inferior wall infarction especially when there is associated right ventricular infarction for fear of causing hyptension and

bradycardia. It is common practice to give oxygen nasally for 24-48 hours to all patients. Beta blockers have been used to limit the size of infarct in early hours because they reduce blood pressure and heart and hence myocardial oxygen demand.

The most important development in the management of Acute Myocardial Infarction in recent times is the concept of reperfusion. Thrombolytics agents must be given to all patients with Acute Myocardial Infarction who reaches hospital within six hours of pain unless contraindicated is associated with limitation of infarct size, improved ventricular function and a low hospital mortality. The agents used for thrombolysis include streptokinase, urokinase, tPA and APSAC. Aspirin should be the first drug given to any patient with Acute Myocardial Infarction.

The other major part of management is that of hemodynamic complications and arrhythmias.

Patients with right ventricular infarction may present with systemic hypotension. Volume loading is the mainstay of treatment. But some patients may not improve with volume loading. In general and initial volume infusion should be used with saline or colloids in 200ml increments in 5-10 minutes. Volume expansion is continued (until hypotension is corrected or the capillary wedge is corrected or the capillary wedge pressure research 15-20mm Hg or right atrial pressure exceeds 15mm of Hg.

If cardiac output is not improved with this carefully administered volume infusion, then dobutamine should be administered to augment right ventricular systolic performance and improve forward cardiac output. The left ventricular ejection fraction also may be improve by dobutamie. Ionootropic agents can increase the oxygen demand of an already ischemic myocardium. But an increase in stroke volume and cardiac index may offset any increase in

myocardial oxygen demand by an augmented myocardial blood flow, produced by an increase in systemic and coronary perfusion pressure.

Administration of crystalloids and or collards will result in maximizing myocardial muscle fibre stretch through volume expansion, upto a certain physiologic limits, creates a greater contractiles force, stronger systolic contraction and improved stroke volume result.

Strategies in Treatment of Right Ventricular Infarction

1. Maintain Right Ventricular Preload

   Volume load-eg. Iv / Saline / Collards

   Although volume loading increase RAP and PCWP, it does not increase cardiac output.

   AV sequential pacing for complete heart block

   Prompt cardioversion for atrial fibrillation.

2. Inotropic Support

Dobutamine is the agent of choice, then adrenaline or Noradrenaline, dopamine.

Dobutamine increases cardiac output, stroke volume index and RVEF, Consequently unloading the right ventricle.

3. Reducing Right Ventricular after load

Intraaortic ballon counterpulsation

Vasodilators (Sodium nitroprusside)

Caution: These may also ↓ LV preload and thus ↓ cardiac output.

4 Reperfusion

Thrombolytic Agents

Direct Angioplasty

5. Temporary Pacing

Temporary pacing should be performed in patients with any of the following:

- Sinus bradycardia unresponsive to drug theraphy

- Mobitz type II second-degree atrioventricular block
- Third-degree heart block
- Bilateral bundle branch block
- Newly acquired bundle branch block
- Right or left bundle branch block in conjunction with first-degree atrioventricular block.

Immediate surgical intervention is often required when PTCA is unsuccessful and patients have persistent chest pain or hemodynamic instability. Patients known to have a mechanical abnormality leading to serve pulmonary congestion or hypotension, such as papillary muscle rupture (with resultant mitral regurgitation) or ventricular septal defect, are candidates for immediate emergency surgery.

AV block is a common complication of right ventricular infarction and usually require temporary passing. AV sequential pacing should be employed in patients with AV block or junctional rhythms.

COMPLICATIONS

1. **Shock**
2. **2nd or 3rd degree heart block – indicates a poor prognosis and occurs in as many as 48% of Right ventricular myocardial infarction.**
3. **Atrial Fibrillation (1/3 of Right ventricular myocardial infarction)**
4. **Ventricular arrythmias**
5. **Ventricular septal rupture – in patients with Right ventricular myocardial infarction plus tranmural posterior septal infarction.**
6. **Right ventricular thrombus formation and subsequent pulmonary embolism.**
7. **Tricuspid regurgitation**
8. **Pericarditis (due to the frequent transmural injury of the relatively thin walled right ventricle)**
9. **Right-to-left shunt through a patent foramen ovale (should be suspected in patients who have hypoxemia that is not responsive to the administration of oxygen).**

AV block is a common complication of right ventricular infarction and usually require temporary pacing. AV sequential pacing should be employed in patients with AV block or junctional rhythms.

If systemic venous congestion is causing significant discomfort to the patient diuretics should be avoided as they can precipitate or aggravate hypotension. Right ventricular function improves with passage of time and systemic venous congestion will usually decrease.

PROGNOSIS

Right ventricular myocardial infarction Occurs in 25-33% patients with inferior wall myocardial infarction. The bedside diagnosis of Right ventricular myocardial infarction is based upon constellation of findings of right sided heart failure including a right ventricular third or fourth heart sounds, elevated jugular venous pressure with a steep 'Y' descent, a positive Kussmaul's sign, arterial hypotension and clear lung by X-ray and physical examination. ST segment elevation of the right sided precordial electrocardigraphic leads, particularly lead V4R, are present in the majority of patients.

Radio nuclide ventriculogrphy and 2-dimensional echocardiography are also sensitive in the detection of right ventricular dysfunction associated with acute MI. Catheterization of the right side of the heart often reveals a distinctive hemodynamic pattern resembling cardiac tamponade or constructive pericarditis.

Rarely Right ventricular myocardial infarction can present as tricuspid regurgitation and rupture of interventricular septum. Shock is observed only in cases with transmural infraction of right ventricle with additional involvement of two or more walls of left ventricle.

Right ventricular infraction with inferior infarction is more susceptible to AV Block pulmonary embolism is said to be more common in right ventricular infraction. Exact incidence of this complication is not clear.

In patients with right ventricular infarction, complete reperfusion of the right coronary artery by angioplasty results in the dramatic recovery of right ventricular performance and an excellent clinical outcome. In contrast unsuccessful reperfusion is associated with impaired recovery of right ventricular function, persistent homodynamic compromise, and a high mortality rate.

- When inferior myocardial infarction is complicated by right ventricular infarction, however, the in-hospital mortality may be as high as 31 percent, as compared with 6 percent of patients with inferior myocardial infarction and no right ventricular involvement.
- Several studies have found that right ventricular dysfunction after a myocardial infraction is an independent risk factor for higher long-term mortality.

- In the vast majority of survivors of right ventricular infarction, manifestations of right ventricular dysfunction return to normal. Clinical and hemodynamic recovery eventually occurs even patients whose right ventricular function remains depressed for weeks or months. This return to normal may be due to the amelioration of concomitant left ventricular dysfunction, resulting in a reduction in right ventricular afterload, or to a gradual stretching of the pericardium with amelioration of its restraining effect.

Finally complete recovery over a period of weeks to months is a rule in a majority of patients suggestions right ventricular stunning rather than irreversible necrosis has occurred.

MATERIALS AND METHODS

This study was conducted during 2006-2007 period. 50 consecutive patients admitted to the coronary care unit or general medical wards with a diagnosis of acute inferior wall infarction were included in the study.

Inclusion Criteria:

Patients having inferior wall myocardial infarction.

All patients included in the study were subjected to ECG examination of V3R and V4R in addition to the conventional 12 leads. Rhythm strip were taken in patients with arrhythmias. ECGs were examined at the time admission, second day and up to the day of discharge.

Only those cases with hyperacute inferior wall infarction were included in the study. Patients with slope elevation of ST segment in leads, II, III and a VF were taken as having hyperacute inferior wall infarction. Right ventricular infarction was diagnosed if there was ST elevation equal to or more than 1 mm in V4R.

All patients were assessed clinically and electrocardiographically with special emphasis on presenting complaints, risk factors, vital signs, arrhythmias and mortality. Patients were followed up till discharge.

Exclusion Criteria:

Patients having history of,

1. Chronic Lung disease
2. Previous MI
3. Rheumatic Heart disease
4. Pericardial disease or LBBB

Because diagnosis of right ventricular infarction is not possible in these cases when ECG is used as the criteria.

Patients who presented after 24 hrs of onset of chest pain were excluded as the ST changes in right ventricular infarction may be transient.

RESULTS & OBSERVATIONS

Observations in 50 patients with acute inferior wall myocardial infarction is presented in this section in descriptive and tabular form.

AGE

Cases were divided into groups of 5 years difference for comparing age and sex incidence. Peak incidence was found in two groups. One group with mean age 42 and another with mean age 62.

TABLE – 1

Age And Sex Distribution Of Patients

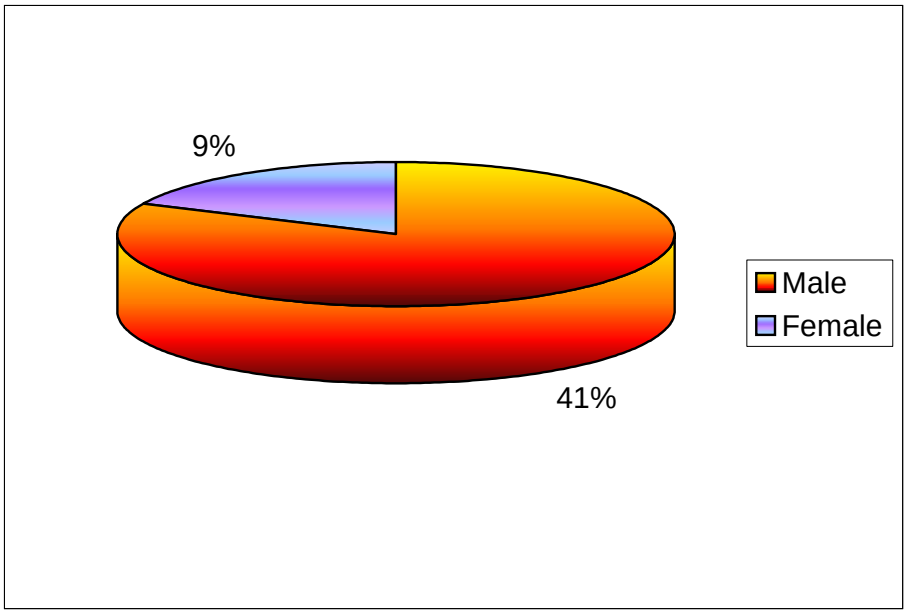
Age	Total No. of Patients	Percentage	Male	Female
30-34	1	2	1	0
35-39	1	2	1	0
40-44	10	20	9	1
45-49	7	14	7	0
50-54	3	6	3	0
55-59	8	16	7	1
60-64	10	20	7	3
65-69	4	8	3	1
70-74	4	8	2	2
75-79	1	2	1	0
80 and above	1	2	0	1
Total	50		41	9

The lowest age was 31 years, who was a male.

The patient with highest age was a female of 80 years of age.

SEX

41 Patients were males and 9 females.



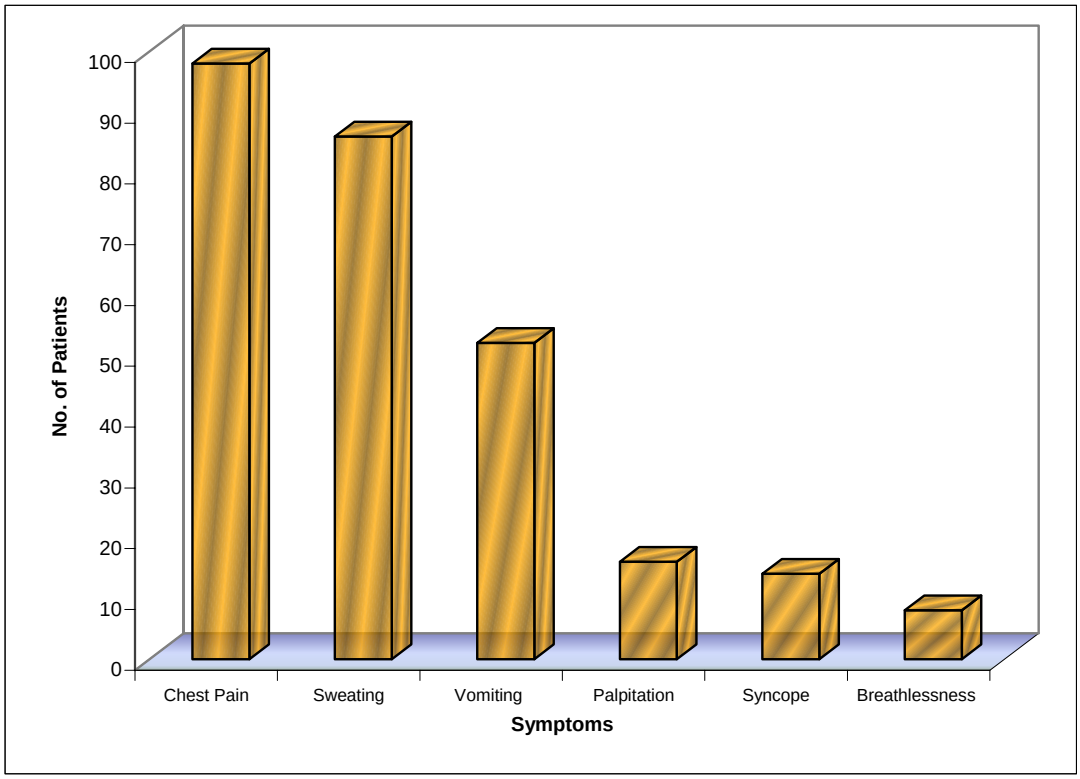
Presenting symptoms (Table 2)

Chest pain: All the patients expect one complain of retrosternal chest pain. The remaining patient had only dyspnoea with no chest pain.

TABLE - 2
Presenting Symptoms

Symptoms	No. of Patients	Percentage
Chest Pain	49	98

Sweating	43	86
Vomiting	26	52
Palpitation	8	16
Syncope	7	14
Breathlessness	4	8



Radiation

Radiation to left upper limb was noticed in 19 (38%) of patients, 3 (6%) patients had radiation to right upper limb. Another 3 patients (6%) had radiation to epigastrium.

Other sites of radiation were neck – 4% Jaw – 2% and left upper limb and left lower limb – 2%.

RADIATION OF CHEST PAIN

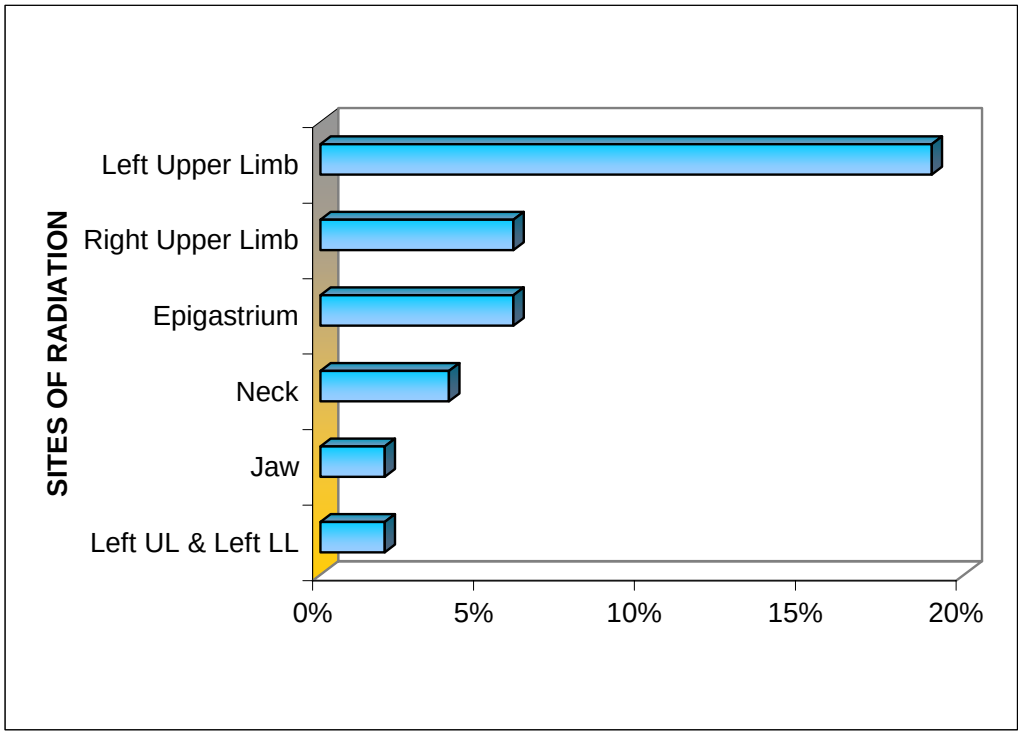
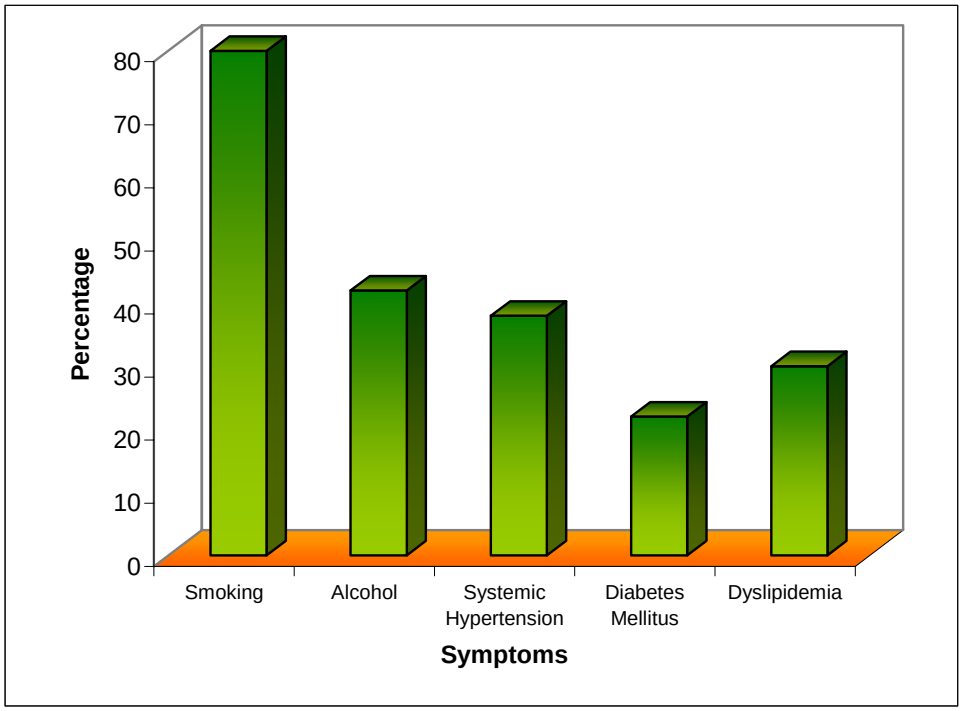


TABLE – 3
Risk Factors

Symptoms	Percentage (%)

Smoking	80
Alcohol	42
Systemic Hypertension	38
Diabetes Mellitus	22
Dyslipidemia	30



Diabetes Mellitus

Was considered to be present when the patients had been told to have the condition, by a doctor. An attempt was made to go through the earlier medical records whenever available. Twelve (22%) patients had diabetes and all were on oral hypglycemics.

Systemic hypertension

Patients were considered to be hypertensive if they had told to had hypertension earlier by a doctor and put on anti hypertensive drugs or serial BP recording in hospital were consistently high. Nineteen (38%) patients had hypertension.

Smoking

Out of 41 males 40 were smokers. None of the female patients were smokers. All the males were smoking 10-20% beedies/cigarettes per day for at least 15-30 years. 82% of them were current smokers.

Clinical examination

Pallor was noticed in 18% of patients had bradycarida. Tachycardia was seen in 6 (12%) patients. Hypotension was observed in 16 patients (32%). 16 patients (32%) had elevated JVP. Right sided third or fourth sounds were detected in 10 (20%) patients. All the patients had evidence of right ventricular infarction in the ECG. Tender hepatomegaly was seen in 2 (4%) patients.

TABLE – 4

Clinical signs at the time of presentation

Clinical Signs	Percentage (%)
Pallor	18
Pulse < 60/mt	32

Pulse > 100/mt	4
BP < 100 mm Hg systolic	32
Elevated JVP	32
Right sided S3/S4	8
Tender hepatomegaly	4

ECG

ECG and clinical criteria for right ventricular infarction were present in 20 (40%) patients.

True posterior wall infarction as evidenced by R/S ratior >1 in VI with upright T wave was observed in 9 (18%) patients. In all these patients the typical change was evident only after 24 hours.

Arrythmias

First degree AV block was observed in 5 (10%) patients.

Second degree – mobitz type 1 block was seen in 3 (6%) patients. Second degree – mobitz type II block was present in 1 (2%) patient.

Complete heart block was noted in 6 (12%) patients.

Transient complete RBBB was noted in 14 cases (28%).

Left anterior hamilock was present in 2 cases (4%)

Most of these arrhythmias were transient requiring no specific treatment, except seven patients who had right ventricular MI,

hypotension and complete heart block who subsequently expired during the hospital stay.

Atrial fibrillation was present in 7 (14%) patients and in both the patients it developed within the first 24 hours.

Papillary muscle dysfunction and mitral regurgitation was noted in 2(4%) patients. This was confirmed by echocardiogram.

Mortality

7 patients expired in the hospital 6 of them within 48 hours of admission. These patients has got right ventricular infarction, profound hypotension, complete heart block and arrythmias.

The remaining one patient developed cerebrovascular accident (right sided temiplegia), CT scan showing infarct in the left middle cerebral artery territory.

TABLE – 5
Complications developed during follow up

Symptoms	Percentage (%)
Atrial Fibrillation	14
I Heart Block	10

II Heart Block	
Mobitz type I	6
Mobitz type II	2
Complete heart block	12
RBBB	28
LAHB	4
VPC's	10
Sinus Bradycardia	10
Mortality	14

DISCUSSIONS

50 proven cases of acute Inferior Wall Myocardial Infarction were selected in the study for analyzing various risk factors and to find out, how many cases of Inferior Wall Myocardial Infarction going for Right ventricular myocardial infarction and to analyse the complications and outcomes of the patients. All patients were admitted with in 24 hrs. of onset of symptoms. All patients were subjected to ECG examination of V_3R, V_4R and Rhythm strip were taken according to the need.

AGE & SEX DISTRIBUTION:

On analyzing the age it was found that acute Inferior wall myocardial infarction is more common above forty years of age. 96% of patients were above 40 among which 83.3% were male.

SUBRAMANIUM et. al. in 2005 had found out median age of Right ventricular myocardial infarction in the Inferior wall myocardial infarction is about 56±13.3 years in the range of 33-83 years.

CHANGTHAN et. al. 1992 found sex distribution in Right ventricular myocardial infarction following Inferior Wall Myocardial Infarction. He says around 80% of cases is of male distribution.

SYMPTOMATOLOGY:

98% of patients presented with chest pain. All of them had typical retrosternal chest pain lasting more than 30 minutes. While analyzing the symptoms other than chest pain the most common was sweating present in 86% patients. It was also associated with cold extremities and pallor.

Vomiting was observed in 52% of patients most of the patients were anxious with a sense of Impending death. Nausea and vomiting

are presumed to be due to stimulation of LV receptor as a part of Bezold-Jarisch Reflex.

14% patients give history of syncope or presyncope of these 7 cases 6 had evidence of Right Ventricular Infarction. High frequency of syncope attack in Right ventricular myocardial infarction is consistent. Hypotension and Brady arrythmias were common in these patients.

MARTIN et. al. in 1989 found 60% of patients with Right ventricular myocardial infarction present as syncope during the time of admission in addition to chest pain.

CLINICAL FEATURES:

On analyzing the clinical features, pallor was noted in 18% of patients. Bradycardia was noted in 28% of patients. Among these 60% were having conduction disturbance and the remainder 40% had associated Right Ventricular Infarction.

In contrast to previous study only 34% of patients had systolic BP less than 100 mm/Hg at the time of presentation. Of these 14 patients (28%) had Right Ventricular Infarction.

Out of 20 patients (40%) with ECG evidence of Right ventricular myocardial infarction, 16 were showing elevated JVP with Positive Kussmaul's sign. Elevated JVP in these cases were exclusively due to Right Ventricular Failure. Previous reports of positive Kussmaul's sign in Right ventricular myocardial infarction varies from (10%-90%) (HARRISON MR et.al).

Those who got Right ventricular myocardial infarction had right sided 3^{rd} or 4^{th} heart sound and 2 patients had tender hepatomegaly.

MARight ventricular myocardial infarctionC et.al. 1990 found that the classical triad of distended neck veins, clear lung fields and hypotension with Bradycardia in 38 (58%) cases of Right ventricular myocardial infarction out of 60 cases.

RISK FACTORS:

On analyzing the Risk Factor for coronary disease, smoking was the most frequent risk factor. In our study 80% were smoker. All of them were males. 42% of the patients were alcoholic. Systemic Hypertension was present in 38% of patients. History of diabetes mellitus was reported by 22% patients. All of them were on oral

Hypoglycemic agent. Dyslipedimia was noted in 15 patients. Of that 5 patients had got diabetes.

BLACKWELL et. al. 2004 found smoking and type 2 Diabetes had increased incidence in Right ventricular myocardial infarction (74%).

ELECTROCARDIOGRAM:

40% of cases showed evidence of Right Ventricular Infarction. All of them showed ST elevation of > 1 mm in V_3R and V_4R. Reported Incidence of Right Ventricular Infarction in patients with Inferior Wall Myocardial Infarction ranges from 25%-53%. MAN JM et.al., ABRAMS DL et.al.

All the patients who had ECG evidence of Right ventricular myocardial infarction had clinical features of the same. The ECG changes reverted to normal or near normal within 24 hrs. of onset of symptoms.

True Posterior Wall Infarction was noted in 9 patients. The classical ECG findings appeared only after 24 hrs. Five of these

patients had associated Right Ventricular Infarction also. Among the five, one patient developed complete heart block on the fist day.

Two out of 9 patients having true posterior wall MI had conduction disturbances. One having complete heart block and the other having second degree Mobitz type II AV Block.

The Recognition of Right Ventricular Infarction varies according to the method by which it is study.

MC GUPTA et.al. in his ECG study directed 27.5% of Right ventricular myocardial infarction in patients with Inferior Wall Myocardial Infarction.

HARMAN O KLIA et.al. in his study with technetium 99m pyrophosphate scintigraphy with ECG found 53% incidence of Right ventricular myocardial infarction in Inferior wall MI.

SINGHAL & LISBANA et.al. 1984 found ECG has 82% sensitivity and 93% specificity for diagnosing Right ventricular myocardial infarction.

ROTH et. al. 1990 found V4R more specific for diagnosing Right ventricular myocardial infarction.

ECHOCARDIOGRAM:

Echo evidence of hypokinesia involving LV Postero-Inferio Wall and lower 1/3rd of interventricular septum was noted in the study of about 27 patients (54%). Of these one patient has got evidence of mitral regurgitation.

SHARPE DN et. al. reported evidence of Hypokinesia involving LV Inferior Wall and lower 1/3rd of interventricular septum following Right ventricular myocardial infarction in 80% of cases.

COMPLICATIONS:

ARRYTHMIAS

The incidence of intraventricular conduction defect in this study was 32%. Of these 28% of cases had RBBB and 4% had LAHB. In this study RBBB appears to be the predominant conduction disturbance.

NEJM April Med. 1998 reported RBBB and complete heart block are the most frequent conduction with abnormalities associated with Right ventricular myocardial infarction.

Complete heart block was present in 12% cases. Of these 6% had Right ventricular myocardial infarction and out of these six patients one expired. All of them improved with conservative management.

Atrial Fibrillation which was transient was noted in 7 patients (14%). This might be due to left Ventricular disfunction or atrial Ischemia. Reported incidence of atrial fibrillation in Acute Myocardial Infarction is 10-15%, but it is only one third in those with Inferior wall MI as compared to Anterior Wall MI.

CARDIOGENIC SHOCK

Out of 50 cases in the study, 48% of cases presented to the hospital with the features of cardiogenic shock. Among the Right Ventricular Infarction 95% of patients had cardiogenic shock during the initial presentation.

BUENOH et. al. reported 90% association of cardiogenic shock those who had Right ventricular myocardial infarction in his study.

R. JOE et. al. also reported 92% association of cardiogenic shock those who had Right ventricular myocardial infarction in his study.

MORTALITY

Only 50% of patients received thrombolytic theraphy and all other were treated conservatively.

There was 7 deaths (14%). Of these 5 patients had Right ventricular myocardial infarction with persistent hypotension and one patient had complete heart block. The other patient developed right sided hemiplegia due to infarction in the left middle cerebral artery.

LOPEZ-PALOP R et. al. Most of the mortality in Right ventricular myocardial infarction is of cardiogenic shock and conduction disturbances.

GNANAVELU, CHOCKALINGAM et. al. reported mortality 16% in the study those who had got Right ventricular myocardial infarction.

The remaining patients left the hospital without major complications.

CONCLUSION

1. The incidence of Right ventricular infarction in this study is about 40%, which is almost equal to that of previous studies.

2. The incidence of Right ventricular myocardial infarction is much higher in males than in females.

3. In males there is a distinct increase in the incidence after the age of 40 years in this study.

4. Almost all patients had typical retrosternal chest pain lasting more than 30 minutes associated with sweating. Syncope was a prominent symptom in patients with right ventricular infarction.

5. Smoking was the most prevalent risk factor.

6. The incidence of True Posterior wall MI in this study was 16%.

7. Hypotension and Bradycardia is a commonest clinical feature associated with Right ventricular myocardial infarction.

8. There should be a strong suspicion of Right ventricular myocardial infarction (98%) in all cases of inferior wall myocardial infarction presenting with features of cardiogenic shock and hypotension.

9. All cases of Inferior wall infarction should have right sided chest leads recorded during ECG examination and this should be done as early as possible. If diagnosis of Right ventricular infarction is correctly made and treated the prognosis is usually good.

10. Mortality is higher in patients with Right ventricular infarction when compared with those without this complication. This was due to higher incidence of conduction disturbances and pump failure. In this study mortality is around 14%.

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PROFORMA

Name Age : Sex :

I.P. NO :

Clo : Time :

1. Chest Pain

2. Perspiration

3. Palpitation

4. Dyspncea

5. Pedal Oedema

6. Syncope

7. Other Symptoms

PAST ILLNESS : SHT / DM / IHD / OTHERS

FAMILY HISTORY :SHT /DM / IHD / SUDDEN DEATH

PERSONAL HISTORY : SMOKING / ALCOHOL / TOBACCO / OTHERS SPECIFY

OCCUPATION : SEDENTARY / NON-SEDENTARY

INVESTIGATIONS

1. Urine – Albumin
   Sugar

   Deposits
2. Blood – Sugar
   Urea
3. Sr – Creatinine
4. Lipid profile

5. Sr. CPK - MB

6. Sr. SGOT

7. ECG

8. ECHOCARDIOGRAM

CLINICAL FEATURE

PULSE BP APICAL IMPULSE

POSITION

CHARACTER

JVP

CVS

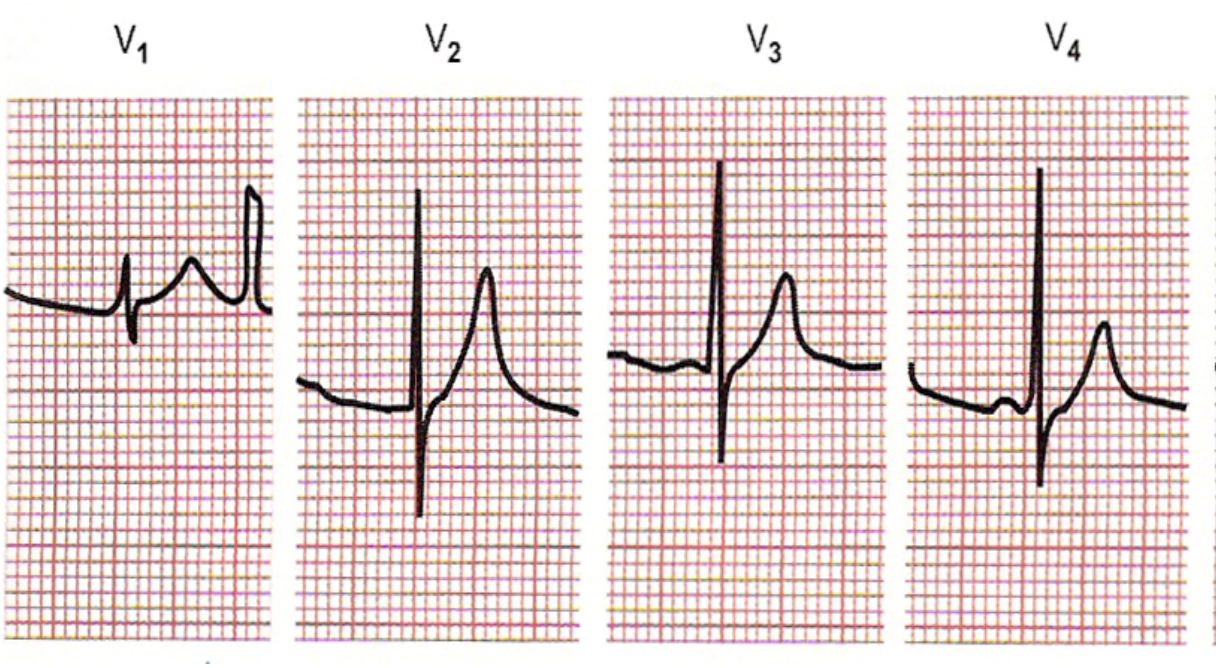
RS

ABDOMEN

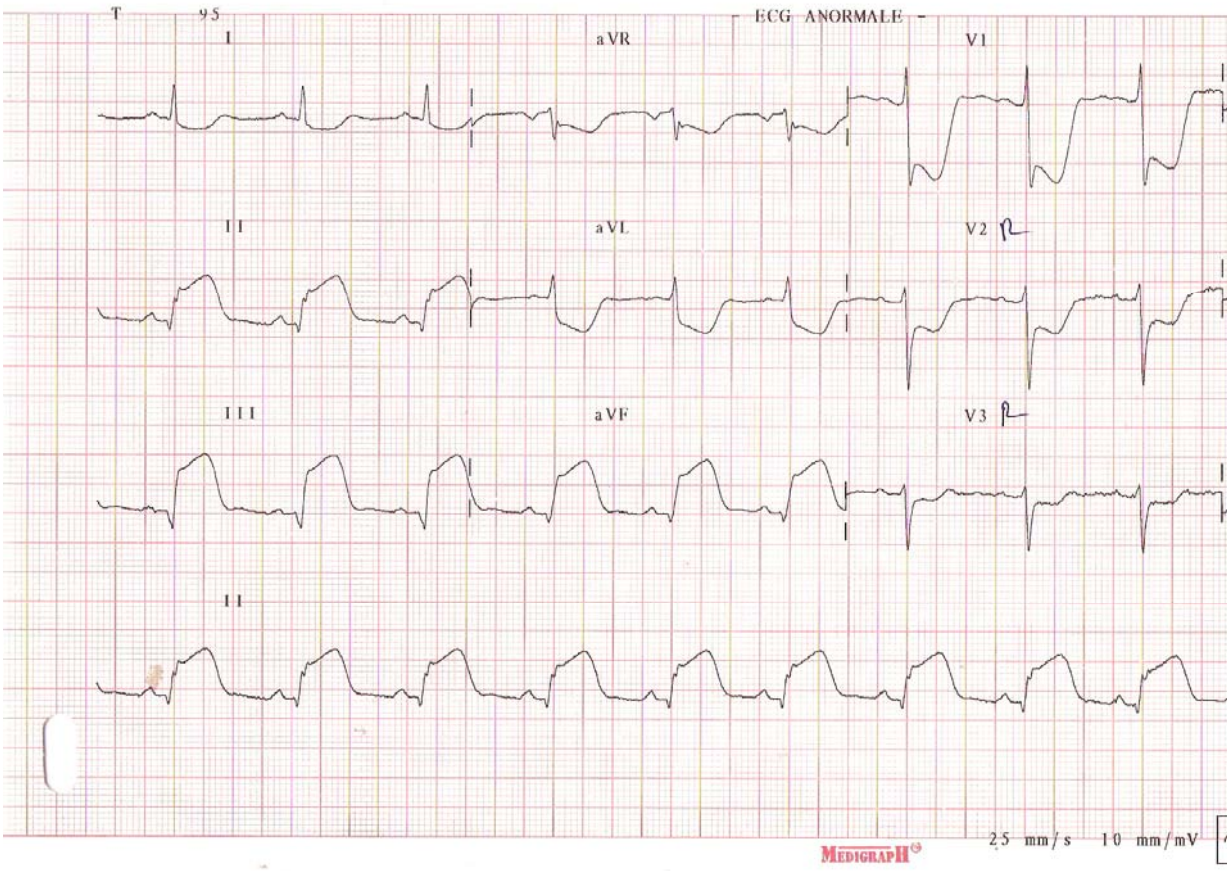
COURSE : CONSERVATIVE TREATMENT /
THROMBOLYSED / ANTICOAGULANTS

COMPLICATIONS DAY OUTCOME

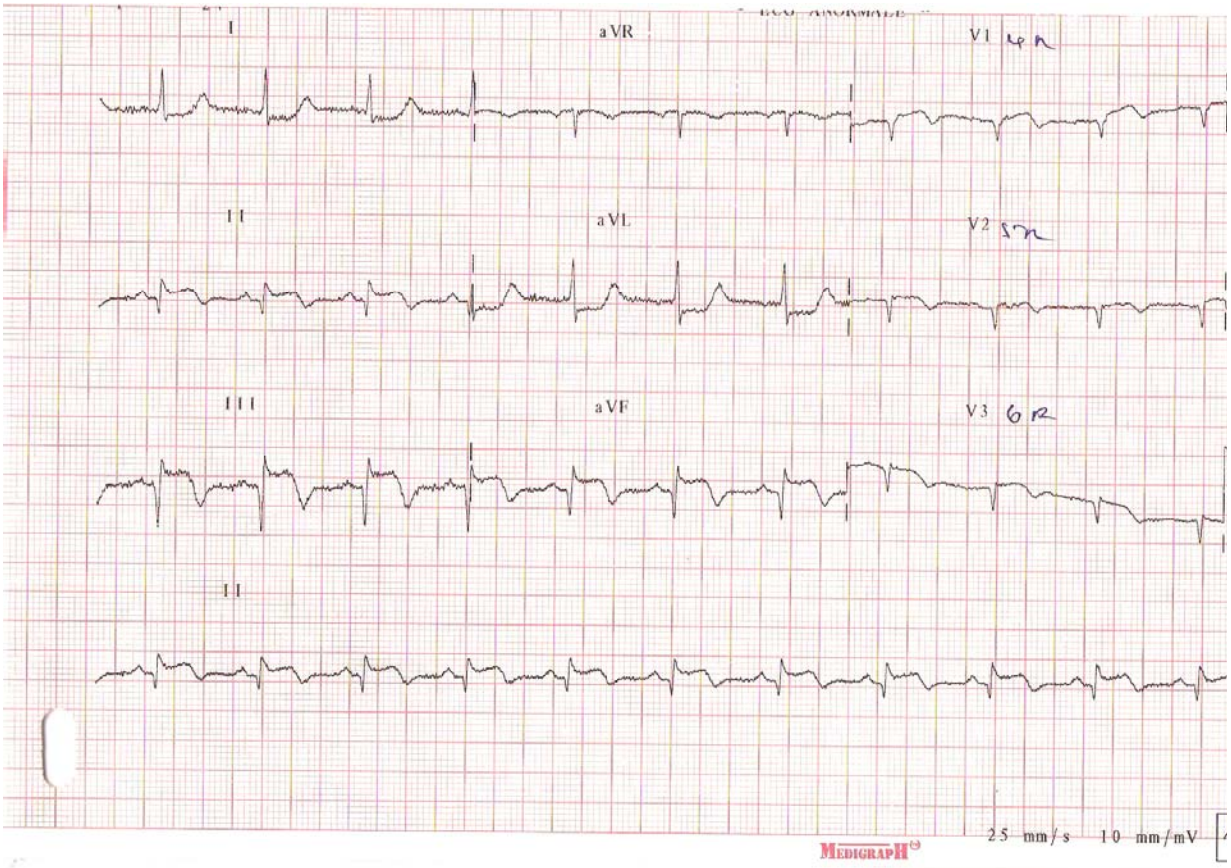
ECG – SHOWING DORSAL INFARCT



ECG – SHOWING INFERIOR WALL MYOCARDIAL INFARCTION



ECG –RIGHT SIDED LEADS RIGHT VENTRICULAR MYOCARDIAL INFARCTION


I
aVR
V1
II
aVL
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MEDIGRAPH
25 mm/s 10 mm/mV